PREFACE

This report summarizes information received from state health departments, medical departments of the Armed Forces, and other sources. It is intended primarily for the use of those with responsibility for disease control activities. Anyone desiring to quote this report should contact the original investigator for confirmation and interpretation.

Contributions to the Surveillance Report are most welcome. Please address them to:

Centers for Disease Control
Attn: Malaria Branch
Division of Parasitic Diseases
Center for Infectious Diseases
Atlanta, Georgia 30333
Telephone: (404) 452-4046
(FTS) 236-4046

SUGGESTED CITATION

Centers for Disease Control: Malaria Surveillance Annual Summary 1985
Issued September 1986

Centers for Disease Control . . . . . James O. Mason, M.D., Dr.P.H., Director

Center for Infectious Diseases . . . . . Walter R. Dowdle, Ph.D., Director

Division of Parasitic Diseases . . . . . Robert L. Kaiser, M.D., Director

Malaria Branch . . . . . Carlos C. Campbell, M.D., M.P.H., Chief

Malaria Surveillance . . . . . Hans O. Lobel, M.D., M.P.H.
Marguerite Pappaioanou, D.V.M., Ph.D.
Kirk D. Miller, M.D.
Alan Greenberg, M.D.
Jacquelin M. Roberts, Statistician

Fluorescent Antibody Laboratory . . . . . Albert Turner
Alexander J. Sulzer, Ph.D.

Publications & Graphics . . . . . Frances H. Porcher, M.A., Chief
J. Philip Thompson, M.S., Writer-Editor

I. SUMMARY

During 1985, 1,045 cases of malaria diagnosed in the United States were reported to the Centers for Disease Control (CDC). This compares with 1,016 cases reported in 1984, an increase of 3%. A total of 444 cases with onset in 1985 were reported in U.S. civilians, more than in any year since the early 1950's. A total of 562 cases were reported in foreigners.

Plasmodium vivax was the parasite identified in 59% of the 1,045 cases, and *P. falciparum* was identified in 29% of the cases. *P. malariae* and *P. ovale* were reported in 5% and 2% of cases, respectively. The species was not determined in the remaining cases.

Only 9 of the 1,045 cases involved persons who acquired the infection in the United States. Congenital infection accounted for 2 cases, and 4 infections were acquired by blood transfusion. In 3 cases the route of infection could not be established.

Twelve deaths attributed to malaria were reported for 1985, compared with 10 such deaths in 1984.

II. TERMINOLOGY

The terminology used in this report is derived from the recommendations of the World Health Organization (WHO)(1). The definitions of the following terms are included for reference purposes.

A. Autochthonous

1. Indigenous--malaria acquired by mosquito transmission in an area where malaria is a regular occurrence.

2. Introduced--malaria acquired by mosquito transmission from an imported case in an area where malaria is not a regular occurrence.

B. Imported

Malaria acquired outside a specific area (the United States, Puerto Rico, and Guam in this report).

C. Induced

Malaria acquired through artificial means, i.e., blood transfusion, common syringes, or malariotherapy.

D. Relapsing

Renewed manifestation (of clinical symptoms and/or parasitemia) of malarial infection, separated from previous manifestations of the same infection by an interval greater than any interval due to the normal periodicity of the paroxysms.

E. Cryptic

An isolated case of malaria not associated with secondary cases as determined by appropriate epidemiologic investigation.

III. GENERAL SURVEILLANCE

A total of 1,045 cases* with onset of illness in 1985 in the United States were reported to the Division of Parasitic Diseases, Center for Infectious Diseases, Centers for Disease Control (CDC); this compares with 1016 cases reported for 1984. Only 31 of the cases occurred in U.S. military personnel. Civilian cases have accounted for the majority of cases each year since 1973 (Table 1).

The number of malaria cases in U.S. civilians increased from 360 cases in 1984 to 446 cases in 1985, a 23.9% increase (Figure 1). Malaria in foreign civilians decreased from 632 reported cases in 1984 to 568 cases in 1985, a decline of 10.1% .

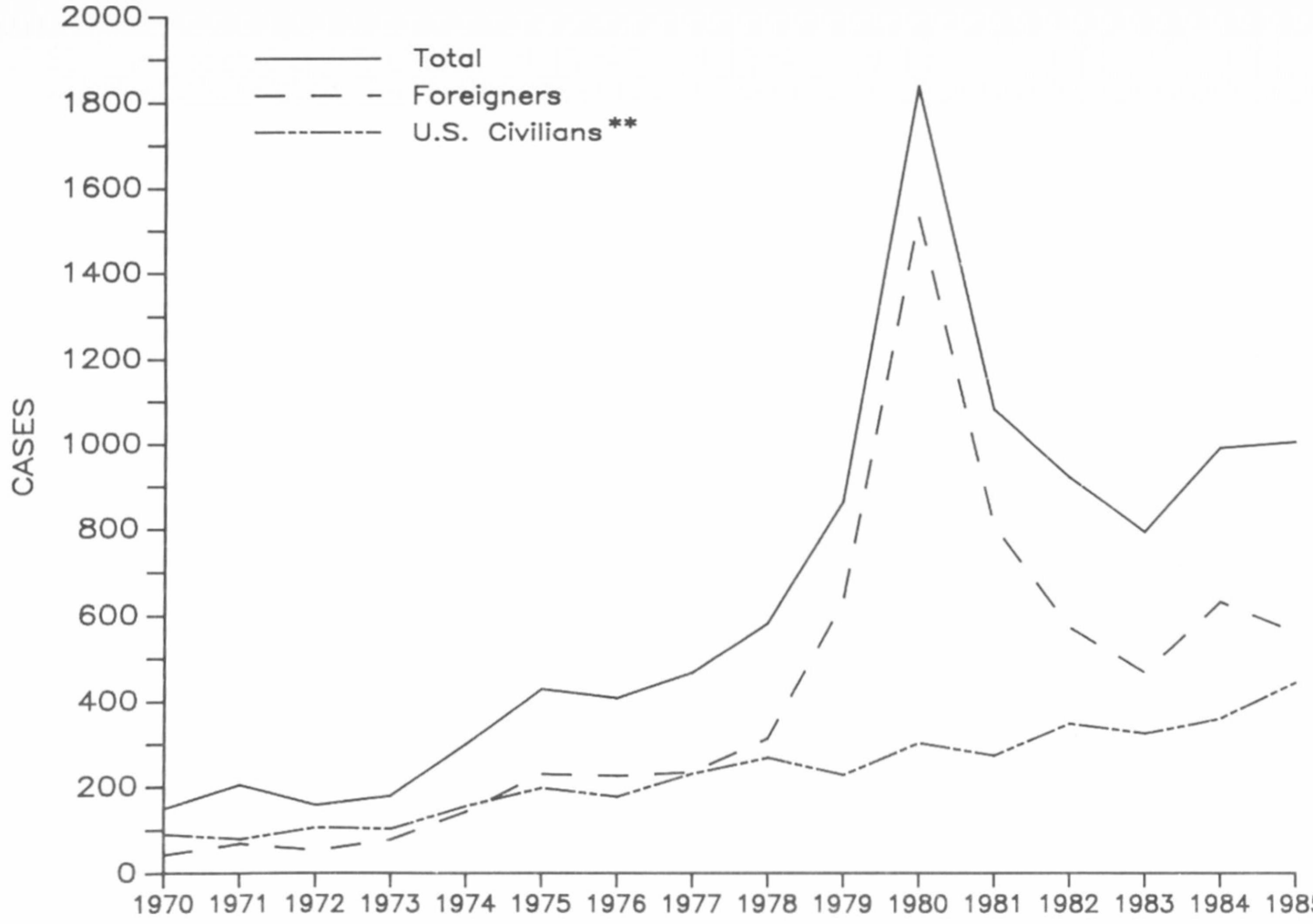
Table 1 All Primary Malaria Cases in Civilians and Military Personnel with Onset of Illness in the United States, 1966-1985*

Year	Military	U.S. Civilians	Foreign Civilians	Unknown	Total
1966	621	89	32	22	764
1967	2,699	92	51	15	2,857
1968	2,567	82	49	0	2,698
1969	3,914	90	47	11	4,062
1970	4,096	90	44	17	4,247
1971	2,975	79	69	57	3,180
1972	454	106	54	0	614
1973	41	103	78	0	222
1974	21	158	144	0	323
1975	17	199	232	0	448
1976	5	178	227	5	415
1977	11	233	237	0	481
1978	31	270	315	0	616
1979	11	229	634	3	877
1980	26	303	1,534	1	1,864
1981	21	273	809	0	1,103
1982	8	348	574	0	930
1983	10	325	468	0	803
1984	24	360	632	0	1,016
1985	31	446**	568	0	1,045

*includes Puerto Rico, the Virgin Islands, and Guam.
**includes 9 cases acquired in the United States.

*A "case" is defined as: 1) an individual's first attack of malaria in the United States, regardless of whether or not he/she had experienced previous attacks of malaria while outside the country, and 2) the presence of a positive peripheral blood smear examined in the local or state health department laboratory. Blood smears from doubtful cases were referred to the National Malaria Repository, CDC, for confirmation of the diagnosis. A subsequent attack in the same person caused by a different Plasmodium species is counted as an additional case. A repeated attack in the same person in this country caused by the same species is not considered to be an additional case.

FIG. 1 CASES OF MALARIA IN U.S. CIVILIANS AND FOREIGNERS, UNITED STATES, 1970–1985*



Only 9 of the 1,045 cases involved persons who acquired the infection in the United States. This was due to congenital transmission in 2 cases, transmission via blood transfusion in 4 cases, and in 3 cases the route of infection could not be established.

The Plasmodium species could be determined in 989 of the 1,045 cases (94.6%). In 1985, P. vivax was identified in 59% and P. falciparum in 29% of the infected individuals (Table 2).

Table 2 All Malaria Cases by Plasmodium Species, United States, 1984 & 1985

Species	1984 Total	1984 Percent	1985 Total	1985 Percent
P. vivax	612	60.2	611	58.5
P. falciparum	261	25.7	303	29.0
P. malariae	53	5.2	49	4.7
P. ovale	20	2.0	21	2.0
Mixed	5	0.5	5	0.5
Undetermined	65	6.4	56	5.4
TOTAL	1016	100.0	1045	100.0

The countries of origin of the 1,045 cases are listed in Table 3.

The geographic distribution of the malaria cases within the United States is shown in Figure 2 by the State in which the patient first developed clinical symptoms of malaria.

Fig.2 GEOGRAPHIC DISTRIBUTION OF MALARIA CASES WITH ONSET IN THE UNITED STATES, 1985

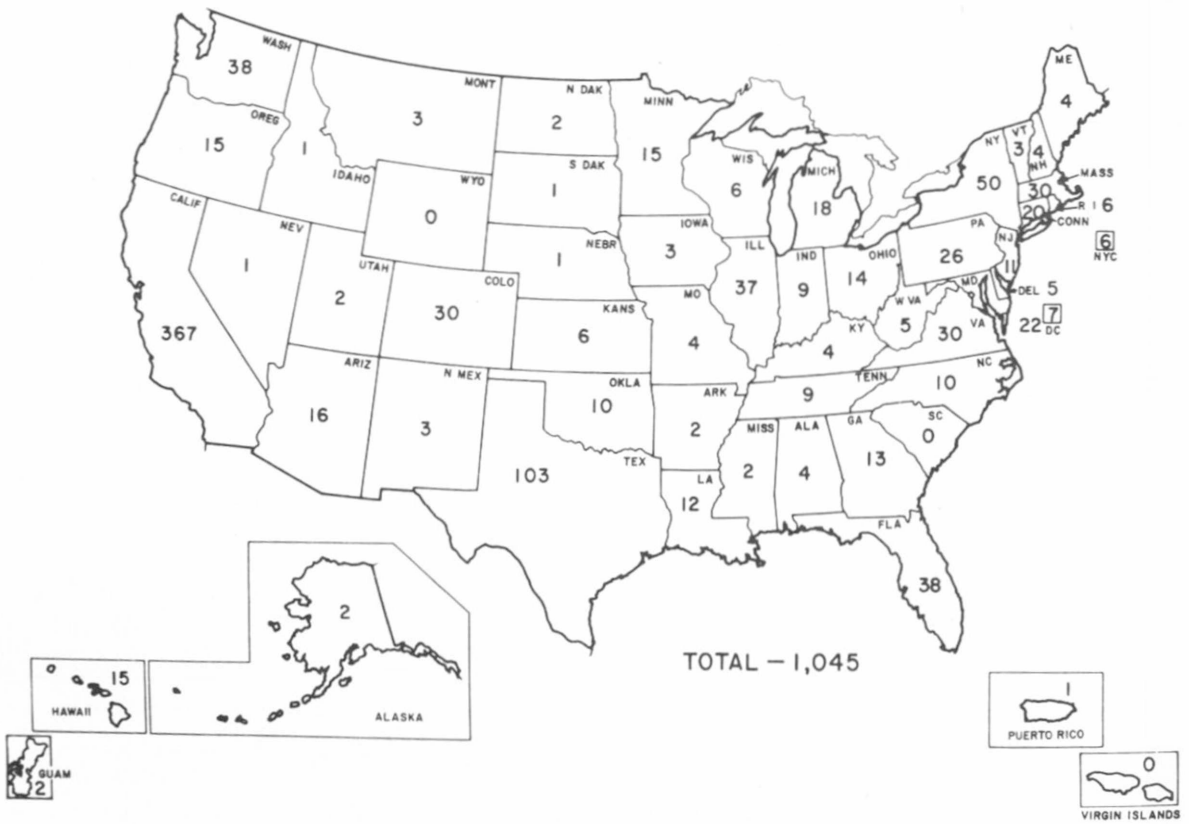


TABLE 3. Malaria Cases by Distribution of *Plasmodium* Species and Area of Acquisition, United States, 1985*

Area of Acquisition	*vivax*	*falciparum*	*malariae*	*ovale*	mixed	unknown	Total
AFRICA	45	218	17	15	1	22	318
Africa, East†	0	5	0	0	0	0	5
Africa, West†	1	4	0	0	0	2	7
Africa, South†	0	1	0	0	0	0	1
Africa, Unspecified†	5	12	1	2	0	3	23
Algeria	1	0	0	0	0	0	1
Angola	0	3	0	0	0	1	4
Benin	0	1	0	0	0	0	1
Burundi	0	2	0	0	0	0	2
Cameroon	0	4	0	1	0	0	5
Central Afr. Rep	0	2	0	0	0	0	2
Chad	0	2	0	0	0	0	2
Congo	0	4	0	0	0	0	4
Ethiopia	3	2	1	0	0	0	6
Gabon	1	2	1	1	0	0	5
Ghana	3	13	0	0	0	1	17
Guinea	0	0	0	1	0	0	1
Ivory Coast	2	1	1	1	0	1	6
Kenya	5	69	1	5	0	4	84
Liberia	2	6	3	0	0	1	12
Malagasy Republic	1	0	0	0	0	0	1
Malawi	0	1	0	0	0	0	1
Mali	0	1	0	0	0	0	1
Niger	0	2	0	0	0	0	2
Nigeria	11	47	7	3	1	6	75
Sierra Leone	0	5	1	0	0	0	6
South Africa	1	1	0	0	0	0	2
Sudan	6	1	0	0	0	0	7
Tanzania	0	9	0	0	0	2	11
Togo	0	0	0	1	0	0	1
Uganda	0	4	1	0	0	0	5
Zaire	3	5	0	0	0	0	8
Zambia	0	6	0	0	0	1	7
Zimbabwe	0	3	0	0	0	0	3
ASIA	305	52	20	3	4	21	405
Asia, South East†	39	19	1	0	0	2	61
Asia, Unspecified†	2	0	0	0	0	0	2
Afghanistan	3	0	0	0	0	0	3
Bangladesh	1	0	0	0	0	0	1
China	1	0	0	0	0	0	1
India	179	22	10	1	0	13	225
Indonesia	13	1	1	0	2	2	19
Kampuchea	0	1	0	0	0	0	1
Malaysia	2	0	0	0	0	0	2
Nepal	1	0	0	0	0	0	1
Pakistan	37	3	5	2	1	2	50
Philippines	18	4	1	0	1	2	26
Saudi Arabia	0	1	0	0	0	0	1
Sri Lanka	0	0	1	0	0	0	1
Thailand	3	1	1	0	0	0	5
Vietnam	6	0	0	0	0	0	6
CENTRAL AMERICA AND CARIBBEAN	120	21	2	0	0	6	149
Central Amer. Unspec.†	6	0	0	0	0	0	6
Belize	2	0	0	0	0	0	2
El Salvador	78	4	1	0	0	1	84
Guatemala	13	0	0	0	0	0	13
Haiti	0	16	0	0	0	1	17
Honduras	18	1	0	0	0	2	21
Nicaragua	3	0	0	0	0	2	5
Panama	0	0	1	0	0	0	1
NORTH AMERICA	110	7	9	1	0	3	130
Mexico	105	5	9	0	0	2	121
United States	5	2	0	1	0	1	9
SOUTH AMERICA	6	2	1	1	0	0	10
America, South†	0	0	1	0	0	0	1
Brazil	2	1	0	1	0	0	4
Colombia	1	0	0	0	0	0	1
Ecuador	2	1	0	0	0	0	3
French Guiana	1	0	0	0	0	0	1
OCEANIA	19	4	1	1	0	3	28
New Guinea	17	3	1	1	0	3	25
Solomon Islands	2	1	0	0	0	0	3
UNKNOWN	4	0	1	0	0	0	5
TOTAL	609	304	51	21	5	55	1045

*Includes Puerto Rico, Virgin Islands and Guam.

†Country Unspecified.

The interval between the date of arrival in the United States and the date of onset of illness was known for 577 of the imported cases for which the infecting Plasmodium species was also identified. Clinical malaria developed within 1 month after arrival in 91.0% of the patients with P. falciparum malaria and in 33.8% of the patients with P. vivax infections (Table 4). Only 14 (2.4%) of the 577 patients became ill 1 year or more after their arrival in the United States.

Table 4 Imported Malaria Cases by Interval between Date of Entry and Onset of Illness and by Plasmodium Species, United States, 1985

Interval (in months)	PLASMODIUM SPECIES									
	vivax	(%)	falciparum	(%)	malariae	(%)	ovale	(%)	Total	(%)
< 1	115	(33.8)	183	(91.0)	13	(54.2)	2	(16.7)	313	(54.2)
1-2	82	(24.1)	10	(5.0)	4	(16.7)	2	(16.7)	98	(17.0)
3-5	63	(18.5)	4	(2.0)	3	(12.5)	3	(25.0)	73	(12.7)
6-11	72	(21.2)	1	(0.5)	3	(12.5)	3	(25.0)	79	(13.7)
≥12	8	(2.4)	3	(1.5)	1	(4.2)	2	(16.7)	14	(2.4)
TOTAL	340	(100.0)	201	(100.0)	24	(100.0)	12	(100.0)	577	(100.0)

Twelve fatal malaria infections were reported in 1985, compared with 10 such infections in 1984. These cases are discussed in Section VII.

IV. MALARIA IN MILITARY PERSONNEL

Thirty-one cases of malaria were reported in U.S. military personnel in 1985. The Army accounted for 11 cases, the Navy for 3, the Air Force for 1, the Marine Corps for 14 cases and for 2 cases the Branch of Service is not known.

V. IMPORTED MALARIA IN CIVILIANS

The number of malaria cases in civilians imported from Mexico approximately doubled from 1984 to 1985 (121 cases in 1985 as compared with 66 in 1984).

Malaria in U.S. citizens accounted for 437 (43.5%) of the 1005 imported cases in civilians, whereas 568 (56.5%) of the cases occurred in citizens of other countries (Table 5). Of the 437 imported cases in U.S. civilians, 220 (50.6%) were acquired in Africa and 101 (23.1%) were acquired in Asia.

Imported malaria in U.S. civilians who had been infected in Africa increased steadily since 1982: 129 cases in 1982, 164 cases in 1983, 170 cases in 1984, and 220 cases in 1985. Of the 220 infections acquired in Africa, 159 (72%) were caused by P. falciparum, and 92 (58%) of these had been acquired in Kenya or Nigeria. P. falciparum infections acquired in Kenya by U.S. citizens increased from 27 cases in 1984 to 67 cases in 1985.

Table 5 Imported Malaria Cases in Civilians, by Area of Infection, United States, 1985

Area of Acquisition	United States		Foreigners		Total	
	Cases	Percent	Cases	Percent	Cases	Percent
Africa	220	50.5	95	16.7	315	31.4
Asia	101	23.0	286	50.4	387	38.5
Central America	25	5.8	99	17.4	124	12.3
Caribbean	13	3.0	4	0.7	17	1.7
Mexico	47	10.8	74	13.2	121	12.1
South America	8	1.8	2	0.3	10	1.0
Oceania	22	0.5	4	0.6	26	2.5
Unknown	1	0.0	4	0.5	5	0.4
TOTAL	437	100.0	568	100.0	1005	100.0

Asia was the area of acquisition of infection in 286 (50%) of the 568 cases in foreign civilians. Infections acquired in India accounted for 156 (27%) of the malaria infections in foreign civilians during 1985.

The principal reasons for foreign travel for imported cases in U.S. civilians are shown in Table 6.

Table 6 Imported Malaria Cases in U.S. Civilians, by Category, United States, 1985

Category	Cases	Percent
Tourist	147	33.6
Business Representative	59	13.5
Government Employee	6	1.4
Missionary	55	12.6
Peace Corps	14	3.2
Seamen/Aircrew	3	0.7
Teacher/Student	21	4.8
Other	46	10.5
Unknown	86	19.7
TOTAL	437	100.0

VI. MALARIA ACQUIRED IN THE UNITED STATES

A. CONGENITAL MALARIA

Two cases of congenital malaria were reported with onset of illness in 1985. Both infections were due to *P. vivax*.

Case 1--On June 14, 1985, *P. vivax* parasites were identified in a blood smear of a 2-month-old infant girl in Houston, Texas, who was hospitalized with severe anemia. The infant received 150 cc of packed red cells. She was treated with chloroquine and had an uneventful recovery. The infant was born in Houston on April 15, 1985. She was jaundiced on April 18, with a total bilirubin of 7.0. She was treated for unspecified sepsis with ampicillin and kanamycin. On May 30, the infant experienced an episode of fever and abdominal extension. She was treated with ampicillin and corticosporin otic drops for possible otitis. No blood smear was taken. On June 14, the infant was admitted to the hospital with fever of 101°F, marked hepatosplenomegaly and severe anemia.

The mother, aged 25 years, immigrated from El Salvador in June 1984. She reported experiencing previous episodes of malaria in El Salvador but had not been treated. She recalled a febrile illness at 15 weeks of pregnancy. A blood smear taken on June 20 from the mother was negative for malaria parasites. Follow-up treatment was not possible.

(Reported by C.E. Alexander, M.D., M.P.H., Director, and Jeffery P. Taylor, M.P.H., Epidemiologist, Infectious Diseases Division, Texas State Department of Health.)

Case 2--A 27-day-old infant girl in New Jersey was admitted on July 1, 1985, with a 3-week history of fever. On admission the infant had splenomegaly and a temperature of 103°F. Examination of a blood smear revealed *P. vivax* parasites. The infant was treated with chloroquine and had an uneventful recovery. The mother had immigrated in January 1985 from El Salvador. She was first seen at the hospital at 32 weeks' gestation. One week later she had an undiagnosed febrile illness. Following the diagnosis of malaria in the infant, her blood smear was examined but malaria parasites were not found. She was treated with chloroquine. Fluorescent antibody titers in sera of the mother and the infant were identical: 1:16384 to *P. vivax*, 1:256 to *P. falciparum* and *P. malariae* and 1:64 to *P. ovale*.

(Reported by K. Davis, M.D., Elizabeth, New Jersey, and the New Jersey State Department of Health.)

B. TRANSFUSION MALARIA

Four cases of transfusion-associated malaria were reported with onset in 1985.

Case 1--A 73-year-old North Dakota woman underwent open heart surgery in April 1985. Three weeks later she developed episodes of fever, chills, and sweating. Malaria parasites were identified in a blood smear. The organisms had characteristics of both *P. ovale* and *P. malariae*. The fluorescent antibody levels were 1:4096 to *P. ovale*, 1:1024 to *P. falciparum*, and 1:64 to *P. vivax* and *P. malariae*. She was treated with chloroquine and had an uneventful recovery.

She had no history of foreign travel or drug abuse, but had received 12 units of packed red cells for the operation. One of the donors was a 29-year-old native from Kenya who had been in the United States continuously for 7 years. He had a history of having had malaria in Africa but denied any febrile episodes since his arrival in this country. His fluorescent antibody titers were 1:262,144 to *P. falciparum*, 1:16,384 to *P. ovale* and 1:256 to *P. vivax* and *P. malariae*.

(Reported by Marvin Cooley, M.D., University of North Dakota School of Medicine, Grand Forks, North Dakota, and James L. Pearson, M.D., North Dakota State Department of Health.)

Case 2--A 41-year-old man who resided in Dallas, Texas, developed episodes of spiking fevers of 103°F on March 24, 1985. Malaria parasites were found in a blood smear. He was treated with chloroquine and had an uneventful recovery. Fluorescent antibody titers were > 1:4096 to *P. falciparum*, 1:1024 to *P. malariae*, 1:256 to *P. ovale*, and < 1:16 to *P. vivax*.

The patient had no history of foreign travel or drug abuse. On March 12 he had undergone coronary bypass surgery and received 3 units of plasma, 5 units of packed red cells, and 20 units of platelets on March 12 and 6 units of packed red cells on March 13. Only one of the donors had a history of foreign travel during the 3 years before donating blood. The serum of this donor was tested for malaria antibodies. These were negative. No blood samples for serologic testing could be obtained from the other donors.

(Reported by Charles Haley, M.D., Houston County Health Department, and Jeffery Taylor, M.P.H., Texas State Department of Health.)

Case 3--A 53-year-old Florida woman developed fever on May 3, 1985, while hospitalized for treatment of leukemia. *P. vivax* parasites were identified in a blood smear. She had no history of foreign travel but had been treated over an extended period with large amounts of packed red cells and platelet concentrates. Malaria fluorescent antibody titers were negative on sera from 14 donors; however, all donors could not be traced.

(Reported by Leo E. Reilly, M.D., St. Petersburg, Florida, Barbara Barrett, M.T., Civitan Regional Blood Center, Gainesville, Florida, and the Florida State Department of Health.)

Case 4--A 65-year-old man residing in San Antonio, Texas, developed chills and fever up to 106°F. on January 1, 1985. On January 15, he was admitted to the Brooke Army Medical Center at Fort Sam Houston. Blood smears showed intracellular organisms, identified as either Babesia or Plasmodium. He was treated with quinine, tetracycline and clindamycin. The malaria antibody titers were >1:4096 to P. falciparum, 1:1024 to P. malariae, 1:256 to P. vivax and P. ovale. Antibody titer to Babesia microti was >1:1024. He underwent a coronary by-pass operation on November 27, 1984, and had received 2 units of packed red cells and 4 units of fresh frozen plasma. The donors of the red cells had both traveled overseas, but results of their serologic tests were negative for Plasmodium and Babesia. No serum samples or travel history could be obtained from the 4 donors of the plasma.

(Reported by Cpt. David J. Looney, M.C., U.S. Army, Fort Sam Houston, Texas.)

C. CRYPTIC MALARIA

Three cases of "cryptic malaria" were reported with onset in 1985. These were isolated cases of malaria not associated with secondary cases, as determined by appropriate epidemiologic investigation. Cryptic malaria is a rare event in the United States: only 2 cases have been reported in the 10-year period from 1975 to 1984, 1 in Louisiana in 1983 and 1 in California in 1981.

Case 1--A 26-year-old woman residing in Dallas, Texas, complained of fever, sore throat, and malaise on June 1, 1986, but her symptoms subsided spontaneously until June 8, when she developed shaking chills, spiking fevers, headache, and diffuse, left-sided pleuritic chest and abdominal pain; she was examined by her gynecologist, who prescribed ampicillin for possible endometritis. On June 12 her temperature rose to 104°F and she was hospitalized with a presumptive diagnosis of tubo-ovarian abscess. A diagnostic laparotomy was scheduled for June 18. On June 17, however, P. vivax parasites were seen on a routine peripheral blood smear, and retrospective examination of slides from June 12 and 14 were positive for P. vivax as well. The diagnosis was confirmed by the laboratory of the Texas State Health Department. The patient was treated with 1.5 g of chloroquine base over 3 days and 14 days of primaquine, and she had an uneventful recovery.

The patient had no history of foreign travel, denied a history of intravenous (i.v.) drug use, and had not received any blood transfusions. In February 1985 she had her ears pierced at a local jewelry store which only used disposable needles. No other cases of P. vivax were reported in Dallas in 1985, and the patient was not aware of anyone with a similar illness. The Dallas Environmental Health Department reported that the nearest mosquito breeding site was approximately 1 mile from her house.

(Reported by Jerry D. Smilak, M.D., D.V. Powell, M.D., St. Paul Medical Center, Dallas, F.W. Becker, M. Ramhdam, City of Dallas Environmental Health Department, Charles Haley, M.D., Dallas County Health Department, and P.V. Fournier, M.P.H., Texas State Department of Health.)

Case 2--A 27-year-old woman residing in Yuba City, California, became ill on August 30, 1985, but did not seek medical attention until 4 a.m. on September 4. P. vivax parasites were identified on a blood smear. Treatment with chloroquine was started on September 4 and with primaquine on September 6. The patient had not traveled outside the United States during the past 6 years, had no history of blood transfusions and denied parenteral drug use. Her latest trip outside the United States was a 14-month trip in 1979 to Western Australia via Guam, Fiji and Samoa (she had not left the plane at these latter locations). None of these areas have endemic malaria. Her only other travel outside the United States was a trip in 1967 to Hawaii, Guam, the Philippines, and Vietnam.

The patient visited a beach area on a river, about 1 mile south of Yuba City, on August 9 from 8 p.m. to dawn and on August 21 from 3 p.m. to 7 p.m. The beach is named Mosquito Beach and is known as a "hangout" for local young persons. It is said to be aptly named because of the large number of mosquitoes there.

(Reported by Joel Hornstein, M.P.H., Sutter County Health Department, Gene Kaufman, Sutter-Yuba Mosquito Abatement District, Robert A. Murray, Dr.P.H., Ronald R. Roberto, M.D., M.P.H., California Department of Health Services.)

Comment: P. vivax malaria is not uncommon among immigrants from India and Pakistan who reside in the area. It is likely that transmission occurred via Anopheles freeborni.

Case 3--This case is reported as Case 11 in Section VII of this report.

VII. DEATHS DUE TO MALARIA IN THE UNITED STATES

Twelve deaths due to malaria were reported. Ten of the infections had been acquired abroad.

Case 1--A 65-year-old Illinois woman spent the Christmas holidays in 1984 abroad: 2 to 3 days in Johannesburg, South Africa; 2 days in Zimbabwe; 3 to 5 days in Kenya on a safari (including overnight stays in a rural area); and 1 to 2 days in Cairo. Throughout this time she took chloroquine weekly for prophylaxis.

The woman returned home on January 2, 1985. Although she felt weak and tired, she attributed this to her recent travel, but 2 days later the woman became confused. On January 8, 1985, she was seen in the emergency room of a local hospital after she had a grand mal seizure. Physical examination revealed hepatomegaly, icterus, and confusion. Results of an emergency computed tomography scan of the head were negative. A blood smear revealed numerous P. falciparum parasites. She was diagnosed to have cerebral malaria.

The patient was started on i.v. quinine 600 mg every 8 hrs., and 3 tablets of pyrimethamine/sulfadoxine via nasogastric tube. Nevertheless, severe hemolysis developed with hemoglobinuria and acute renal failure. The patient died within 24 hours of the diagnosis after receiving 2 doses of i.v. quinine and 1 dose of pyrimethamine/sulfadoxine. No autopsy was performed.

(Reported by John Matseshe, M.D., Libertyville, Illinois, and the Illinois State Department of Health.)

Case 2--On March 29, 1985, a 30-year-old California man was admitted to a local hospital after experiencing a seizure. He had a temperature of 106°F. On April 1, parasites identified as P. malariae were noted on a blood smear. He was treated with 1500 mg of chloroquine base over 3 days but his hemoglobin levels and urine output declined. On April 3 the patient became comatose and died on April 30 without regaining consciousness. Subsequent examination of the initial blood slides by the San Bernardino Public Health Laboratory and the State's Microbial Diseases Laboratory revealed P. falciparum. No autopsy was performed.

The patient had traveled from February 15 to March 15, 1985, to a village 100 miles north of Bombay. He returned to California on March 22. He had not taken malaria prophylaxis. Chloroquine-resistant P. falciparum has been reported in that part of India, but no follow-up slides were available to determine the parasitological responses to the chloroquine, nor could blood drug levels be determined.

(Reported by Alex F. Taylor, M.P.H., and G.R. Pettersen, M.D., M.P.H., San Bernardino County Department of Public Health, Robert A. Murray, Dr.P.H., and Ronald R. Roberto, M.D., M.P.H., California Department of Health Services.)

Case 3--A 29-year-old woman residing in Contra Costa County, California, had traveled in East Africa for 4 months and returned on April 6, 1985. She had spent most of the time in Kenya and Tanzania but had not taken chemoprophylaxis because of religious convictions. She experienced intermittent fevers during the return travel. A home-care nurse was contacted on April 11, but the patient was unresponsive and pronounced dead on arrival at the hospital. Autopsy revealed hepatosplenomegaly, pulmonary congestion and edema. P. falciparum parasites were present in the myocardium, lungs, adrenal glands, cerebellum, cerebrum, spleen, thyroid, kidneys and liver. Malaria pigment was seen in the liver, kidney and spleen.

(Reported by Barbara Benda, P.H.N., Contra Costa County Department of Health, Louis E. Dougherty, M.D., Contra Costa County Coroner's Office, Martinez, California, Edward K. Markel, M.D., Kaiser Foundation Medical Center, Oakland, California, Robert A. Murray, Dr.P.H., and Ronald R. Roberto, M.D., M.P.H., California Department of Health Services.)

Case 4--On March 4, 1985, a 78-year-old California woman consulted a physician because of generalized weakness. She was afebrile then, but developed fever and malaise on March 6. On March 11 she became confused and was brought to the hospital. She was noted to be lethargic, febrile and jaundiced. Blood smear examination revealed P. falciparum rings and gametocytes. The patient initially improved when treated with oral quinine, pyrimethamine and sulfadiazine. She subsequently deteriorated and became progressively confused, somnolent and comatose. She developed acute respiratory and renal failure. On March 19, malaria parasites were no longer detectable, but the patient died on March 21.

The patient had returned to California on February 26, 1985, from a 2-week tour of game parks in Kenya. She took weekly chloroquine/primaquine tablets during her trip and continued her prophylaxis through the first week of March.

(Reported by L.O. Oslund, M.D., David Hissey, M.D., Sue Hunt, R.N., San Diego County Health Department, and Robert A. Murray, Dr.P.H., California Department of Health Services.)

Case 5--A 49-year-old native from Sri Lanka, who worked in Nigeria for a U.S. oil company, developed fever while in Florida on April 3, 1985. Although the illness was initially diagnosed as a cold, P. falciparum was daignosed on April 6. He had developed renal failure and was treated with i.v. quinine. The patient died on April 11. He had not used chemoprophylaxis while in Nigeria.

(Reported by N. Lopez, M.D., Boynton Beach, Florida, and the Florida State Health Department.)

Case 6--A 62-year-old Massachussetts man complained of muscle spasms on March 1, 1985, for which he was treated with muscle relaxants. Seven days later he was admitted to the hospital with a fever of 104°F. The next day P. falciparum parasites were identified in a blood smear. Treatment with quinine, sulfadiazine and pyrimethamine was promptly instituted. The parasitemia responded, but he developed adult respiratory distress syndrome and died on April 13. Autopsy revealed marked pulmonary fibrosis, pulmonary congestion, bronchopneumonia, and an enterobacter lung abscess.

The patient had traveled to Kenya as a tourist from February 2 to 18, 1985. He had taken weekly chloroquine and Fansidar without interruption from January 19 to February 25. Pre-treatment serum samples showed chloroquine and sulfadoxine levels consistent with adequate chemoprophylaxis.

(Reported by Richard Glew, M.D., Worcester, Massachussetts, and the Massachussetts Department of Health.)

Case 7--A 40-year-old Haitian man who resided in New Jersey visited Haiti from October 4-8, 1985. He did not take chemoprophylaxis. On October 18, 1985, he experienced fever and chills; on October 21 he became delirious, and was brought to the emergency room. Blood smear examination showed a 6% P. falciparum parasitemia. Therapy with intramuscular chloroquine phosphate, 250 mg, was initiated. He was received an infusion of 10 mg/kg of quinidine over 1 hour, followed by a continuous infusion at the rate of 0.02 mg/kg/min. Despite clearance of the parasitemia within 48 hours, progressive renal, neurological, and pulmonary failure ensued, and the patient died on October 24.

(Reported by F. Yen, M.D., Elizabeth, New Jersey, and the New Jersey State Department of Health.)

Case 8--A 55-year-old woman had worked as a missionary in Haiti for the past 10 years, returning to the United States periodically for visits. She took no malaria chemoprophylaxis while in Haiti. She returned to the United States on September 28, 1985. On October 3, 1985, she developed a fever and chills; on October 5 she experienced fever and diarrhea, and on October 8 became confused. She refused medical attention and remained at home in a deteriorating condition for the next 10 days. On October 18 her family noted that she had stopped breathing and called an ambulance. She was pronounced dead on arrival at the hospital. Post-mortem examination revealed numerous P. falciparum parasites in the blood, spleen, and liver.

(Reported by Fred B. Jordan, M.D., Chief Medical Examiner, Oklahoma City, Oklahoma, and Gregory R. Istre, M.D., Oklahoma State Department of Health.)

Case 9--On November 15, a 75-year-old Tennessee man was admitted to a hospital with a 1-week history of not feeling well. Two days before admission he developed a fever. On admission he was confused and slightly dehydrated and hypotensive. The patient had recently returned from a business trip to Ghana and had not taken chemoprophylaxis for malaria. A blood smear showed that 3% of the red blood cells were infected with P. falciparum. He was treated with oral quinine, 650 mg tid. On November 16 the parasitemia was 0.9%, and no parasites were found by the morning of November 17. The patient developed a lower lobe infiltrate, and his neurologic and hemodynamic state deteriorated. He died on November 17.

(Reported by W.R. Mixen, M.D., Knoxville, Tennessee, and the Tennessee State Department of Health.)

Case 10--On December 22, 1984, a 51-year-old minister returned to Michigan after a 2-week trip to Liberia. He took no malaria chemoprophylaxis. On January 4, 1985, 13 days after his return to Michigan, he developed fever, chills, nausea, and vomiting, but he thought he had the "flu" and did not seek medical attention. His symptoms persisted and on January 11 a physician was consulted. P. falciparum parasites were identified in a blood smear, and the patient was admitted to a local hospital. He was treated with oral chloroquine and seemed to improve initially; on the morning of January 13, however, he became comatose and died.

(Reported by Rupert Edwards, M.D., Detroit, Michigan, and Melinda Love, M.D., Detroit City Health Department, and the Michigan State Department of Health.)

Case 11--A 73-year-old male patient in a hospital in Boston had been admitted on November 15, 1985, for a femoral-popliteal bypass operation. His hospital course was complicated by pneumonia caused by gram-negative organisms and a cardiac arrest with ventricular fibrillation on December 18. On December 25, a hematology laboratory technician noted a P. falciparum parasitemia with a parasite density of more than 50% on a routine blood smear. The patient was hypotensive and comatose, and he was treated with pressor agents and 300 mg of i.v. quinidine. Later he received a second dose of 300 mg of i.v. quinidine, followed by a 1 mg/min drip. He received an exchange transfusion with 6 units of packed red cells, and 3 units of whole blood were phlebotomized manually. Despite these measures the patient died on December 26.

The patient had last traveled abroad to the Philippines in 1945. He had no history of malaria or drug abuse. He had received 5 units of packed red cells before December 25: 2 units on December 3, 2 units on December 11, and 1 unit on December 21. Retrospective examination of blood slides stored in the laboratory revealed the presence of P. falciparum as early as December 13. All 5 donors were contacted and denied any history of recent travel to malaria endemic areas. Blood smears of all donors were negative. Sera of all donors were negative for anti-malarial antibodies by indirect immunofluorescence tests. The patient's serum had a titer of > 1:65,536 to P. falciparum and < 1:16 to P. vivax, P. ovale and P. malariae.

During 1 day of his hospitalization in the intensive care unit (ICU), another patient who was from Ethiopia, had been treated for P. falciparum in the same ICU, 2 beds away from the patient. Cross-contamination of blood between the 2 patients, however, could not be established.

(Reported by L. Mofferson, M.D., R. Lawrence, M.D., Cambridge City Hospital, Boston, Massachussetts, M. Popovsky, M.D., Director, Northeast Region, American Red Cross, Boston, Massachussetts, J. Harris, M.D., and G. Grady, M.D., Massachussetts Department of Health.)

Case 12--A 78-year-old California woman was admitted on November 23, 1985, to a Los Angeles hospital; she was comatose, with renal and hepatic failure. P. falciparum malaria was diagnosed on the basis of a blood smear examination. The patient died shortly after the diagnosis was made and before specific treatment could be initiated. The patient had returned 7 days before admission from tourist travel to Kenya. During that week she had experienced myalgia, fever, chills, sweats and lethargy. Chloroquine prophylaxis had been prescribed, but it is not known if she had taken this medication.

(Reported by Michael P. Tormey, M.P.H., Los Angeles County Department of Health Services, Frances Pincus, M.D., Maurice Zagha, M.D., Los Angeles, California, and Robert A. Murray, Dr.P.H., California Department of Health Services.)

VIII. PREVENTION OF MALARIA

Guidelines for the prevention of malaria in travelers have been published by the Center for Prevention Services, CDC, in "Health Information for International Travel 1986," June 1986, HHS publ. no. (CDC) 86-8280. This booklet also provides information about countries and, where applicable, areas within each country, where malaria risk exists. In addition, areas in the world where chloroquine-resistant strains of P. falciparum are known to exist are listed. The booklet is available from Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402.

IX. MICROSCOPIC DIAGNOSIS OF MALARIA

Early diagnosis of malaria requires a high level of clinical suspicion and, in particular, a comprehensive travel history taken from every patient with a fever of unknown origin. Once malaria is suspected, a Giemsa-stained smear of peripheral blood should be examined for the presence of parasites. Since the accuracy of diagnosis is dependent on the quality of the blood film, the following guide is offered for the proper preparation of thick and thin blood smears.

1. Manufacturers' "pre-cleaned" slides are not considered clean enough for use in malaria diagnosis. Before use, wash these slides in mild detergent, rinse them thoroughly in warm running water, then in distilled water, and dip them in ethyl alcohol (90%-95%). Then, wipe slides dry with a lintless cloth or tissue for immediate use or store them in 95% alcohol until needed.

2. Clean the patient's finger with alcohol and wipe the finger dry with a clean cloth or gauze.

3. After puncturing the finger with the blood lancet, allow a large globule of blood to form.

4. Place the cleaned surface of the slide against the drop of blood and, with a quick circular motion, make a film the size of a dime in the middle third of 1 end of the slide. Ordinary newsprint should be barely legible through such a wet drop (Figure 3). (Excessive mixing or stirring with a second slide leads to distortion of blood cells and parasites.)

5. Wipe the finger dry and gently squeeze a small drop of blood from the puncture, placing it at the edge of the middle third of the same slide (Figure 4).

6. Apply a clean "spreader" slide to the edge of the small drop at a 45° angle and allow the blood to extend about two-thirds of the slide width; then, keeping even contact, push the spreader forward along the slide. This will produce an even layer of red blood cells with a "feathering" at the lower edge (Figure 5).

7. While the thick blood film dries (minimum of 6 hours at room temperature)**, keep the film horizontal and protected from dust and insects.

8. Label the slide in the upper part of the thin film with the date and the name or initials of the patient as illustrated (Figure 5).

**If a rapid diagnosis is desired, make the thick and thin films on separate slides. The thin film can be air dried, fixed with methyl alcohol and stained immediately. If no parasites are found on the thin film, examine the thick film for organisms not detected on the thin preparation.

Fig. 3

in all their phases. The importance of the examination of blood films for the presence of malaria parasites will be fully understood

Fig. 4

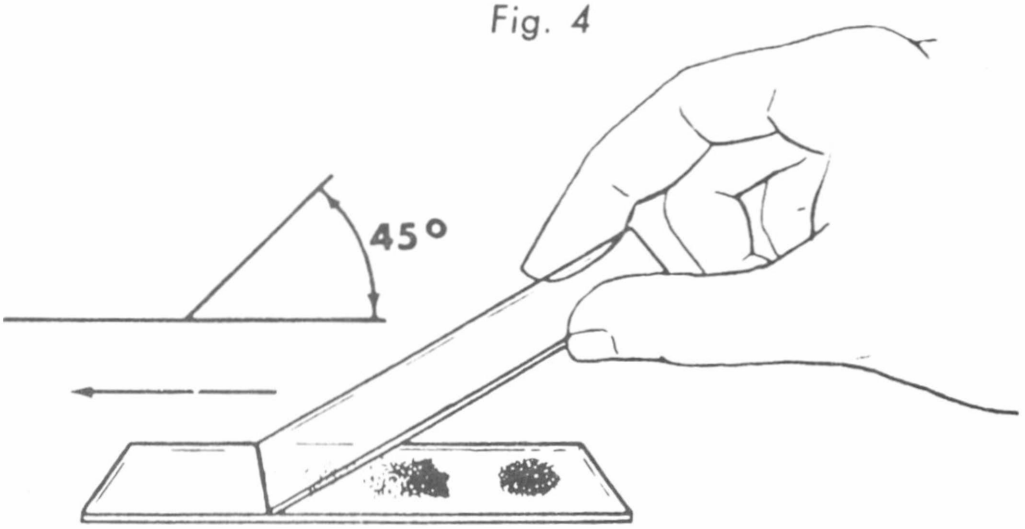
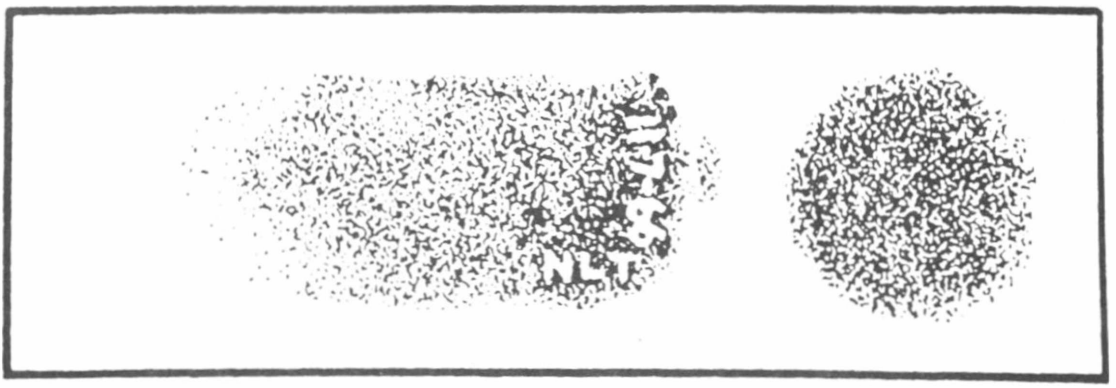


Fig. 5



ACKNOWLEDGMENT

The Malaria Surveillance Report, prepared annually at the Centers for Disease Control, is based on information provided in individual case reports. excellent support given to malaria surveillance by state and local health departments and personnel of the preventive medicine services of the U.S. Army Navy, and Air Force is greatly appreciated.

REFERENCE

1. World Health Organization. Terminology of malaria and of malaria eradication, 1963, World Health Organization, Geneva, p 32.

STATE AND TERRITORIAL EPIDEMIOLOGISTS

Key to all disease surveillance activities are the State and Territorial Epidemiologists. Their contributions to this report are gratefully acknowledged. The persons listed were in the positions shown as of March 1, 1986.

Alabama	Wallace E. Birch, DVM
Alaska	John P. Middaugh, MD
Arizona	Glyn G. Caldwell, MD
Arkansas	Thomas C. McChesney, DVM
California	James Chin, MD
Colorado	Stanley W. Ferguson, MD
Connecticut	James C. Hadler, MD
Delaware	Paul R. Silverman, DrPH
District of Columbia	Martin E. Levy, MD
Florida	E. Charleton Prather, MD
Georgia	R. Keith Sikes, DVM
Hawaii	Arthur P. Liang, MD, MPH
Idaho	Charles D. Brokopp, DrPH
Illinois	Byron J. Francis, MD
Indiana	Charles L. Barrett, MD
Iowa	Laverne A. Wintermeyer, MD
Kansas	Robert French, Acting
Kentucky	M. Ward Hinds, MD
Louisiana	Joyce B. Mathison, MD
Maine	Kathleen F. Gensheimer, MD
Maryland	Ebenezer Israel, MD
Massachusetts	George F. Grady, MD
Michigan	Kenneth R. Wilcox, Jr., MD
Minnesota	Michael Osterholm, PhD, MPH
Mississippi	Fred E. Thompson, MD
Missouri	H. Denny Donnell, Jr., MD
Montana	Judith K. Gedrose, MD
Nebraska	Paul A. Stoesz, MD
Nevada	George Reynolds, MD, Acting
New Hampshire	Eugene Schwartz, MD
New Jersey	William E. Parkin, DVM
New Mexico	Harry F. Hull, MD
New York State	Dale Morse, MD
New York City	Stephen Schultz, MD
North Carolina	J.N. MacCormack, DVM
North Dakota	James L. Pearson, MD
Ohio	Thomas J. Halpin, MD
Oklahoma	Gregory R. Istre, MD
Oregon	Laurence Foster, MD, MPH

Pennsylvania	Ernest J. Witte, MD
Puerto Rico	Jose G. Rigau, MD
Rhode Island	Richard A. Keenlyside, MBBS
South Carolina	Richard L. Parker, DVM
South Dakota	Kenneth A. Senger
Tennessee	Robert H. Hutcheson, Jr., MD
Texas	Charles E. Alexander, MD, Acting
Utah	Craig R. Nichols, MPA
Vermont	Richard L. Vogt, MD
Virginia	Grayson B. Miller, Jr., MD
Virgin Islands	John N. Lewis, MD
Washington	John M. Kobayashi, MD
West Virginia	Loretta E. Haddy, MS
Wisconsin	Jeffrey P. Davis, MD
Wyoming	Harry C Crawford, MD
Guam	Robert L. Haddock, DVM
Ponape*	Eliuel K. Pretrick, MO, MPH
Marshall Islands	Isao Kisino
Northern Mariana Islands*	Jose T. Villagomez, MO
American Samoa	Julia L. Lyons, MD, MPH
Palau*	Anthony H. Polloi, MD, Acting

* Formerly Trust Territory of the Pacific Islands

☆U. S. GOVERNMENT PRINTING OFFICE: 1986, 730-009/45648